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True Uncomplicated Obesity Is Not Related to Increased Left Ventricular Mass and Systolic Dysfunction

I found the recent study by Peterson et al. (1) to be quite interesting. They found that obesity in young and otherwise healthy women is associated with increased left ventricle (LV) mass, LV concentric remodeling, and decreased systolic and diastolic function. I applaud that LV function and diastolic and systolic functions were obtained by use of second harmonic imaging and tissue Doppler imaging, a highly sensitive and specific echocardiographic technique. This procedure allowed the investigators a very accurate evaluation of LV parameters. Nevertheless, I have some comments on the study design and the results.

The first and main criticism is related to the inclusion criteria that they applied to define "healthy" obese women. The researchers put fasting glucose <126 mg/dl or a glucose level <200 mg/dl 2 h after an oral glucose tolerance test (OGTT) and total cholesterol level <260 mg/dl and/or triglyceride level <400 mg/dl as cut-off points to exclude the presence of diabetes mellitus and dyslipidemia, respectively. These parameters do not fill the Adult Treatment Panel III guidelines for cardiovascular risk factors (2). According to the criteria applied in the Peterson et al. (1) study, it is also not possible to exclude the presence of impaired fasting glucose and glucose intolerance assessed by fasting glucose between 110 and 125 mg/dl and glucose <140 mg/dl 2 h after an OGTT, respectively (3). The cut-off point of 400 mg/dl for triglycerides is not acceptable to define a normal plasma lipid profile. In addition, the investigators do not report data on high-density lipoprotein cholesterol (HDL-C) and low- density lipoprotein cholesterol, widely recognized as cardiac risk factors. Hence, there is no evidence that these obese women are healthy subjects.

The presence of underevaluated co-morbidities could explain the finding of increased LV mass and diastolic and systolic dysfunction in these obese women. In fact, the impact of impaired fasting glucose, glucose intolerance, low HDL-C, and hypertriglyceridemia on LV parameters is well known.

We agree with Peterson et al. (1) that the effects of obesity on cardiac structure and function in young and otherwise healthy subjects are still debated. We have performed an investigation on the influence of excess fat only on cardiac morphology and function (4). That study showed that obesity per se, although a disease of long duration, in absence of glucose intolerance, impaired fasting glucose, diabetes, hypertension, and dyslipidemia, is associated only with an impairment of diastolic function and hyperkinetic systole and not with LV hypertrophy. Obese subjects without any co-morbidities, called "uncomplicated" obese subjects, present left atrium and aortic root enlargement as consequences of impaired diastole and hyperdynamic systole. Hyperkinetic systole, estimated by both endocardial and midwall parameters, seems to be due to the increased cardiac work necessary to supply a high oxygen consumption for the large body surface of obese subjects. We have not found increased LV mass or true LV hypertrophy in uncomplicated obese subjects, and insulin resistance seems to be a determinant of LV mass stronger than body mass index (5). Nevertheless, this issue is still controversial.

In addition, obese subjects showed an increase of preload, probably also due to hyperinsulinemia and its sodium retentive action, leading to the development of LV eccentricity more than LV concentric remodeling.

The very restricted inclusion criteria that we applied in our studies in defining "uncomplicated obesity" allowed us to estimate the influence of obesity per se on LV parameters.

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